ECHOCARDIOGRAPHIC EVALUATION OF CARDIAC STATUS IN PATIENTS OF SYSTEMIC HYPERTENSION

THESIS FOR

DOCTOR OF MEDICINE

(INTERNAL MEDICINE)

BUNDELKHAND UNIVERSITY

JHANSI (U.P.)

2006 DEEPAK LALHARIA

Dedicated

To

Respected

Teachers, Parents,

Friends & my Supportive

Family members

CERTIFICATE

This is to certify that the work entitled "*Echocardiographic evaluation of cardiac status in patients of systemic hypertension*" which is being submitted as thesis for M.D. (Medicine) Examination 2006 of the Bundelkhand University, Jhansi, has been carried out by **Dr. Deepak Lalharia** in the Department of Medicine, M.L.B. Medical College, Jhansi.

This method described was undertaken by the candidate himself and the observations recorded have been periodically checked up. He has put in the necessary stay in the department as per university regulations, and has fulfilled the conditions required for the submission of the thesis according to the University regulations.

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Deepak Lalharia

Date : 26/10/05 Dr. Deepak Lalharia

CONTENTS

Introduction

INTRODUCTION

Systemic hypertension is now known as an important public health problem in developed and developing countries with changing life style and increasing longevity. It is a common, asymptomatic, easily detectable, usually easily treatable disease yet, it leads to lethal complications as in majority it is either untreated or is inadequately treated. Uncontrolled BP is attributed to its inherent propensity to induce vascular damage, leading to cardiovascular, cerebral, renal and ophthalmic complications.

Although diastolic dysfunction is the earliest evidence of involvement of heart in hypertension. This is not pathognomic of hypertensive heart disease, as similar changes may be present in aged persons or patients having coronary artery disease, unrelated to hypertension. Left ventricular hypertrophy (LVH) is therefore considered as a hall mark of hypertensive heart disease, as systolic dysfunction usually appears late in course of disease. Patients with left ventricular hypertrophy have increased risk of angina pectoris, acute coronary syndrome, ventricular arrhythmias, sudden cardiac death (SCD) and congestive cardiac failure. Thus

LVH has emerged as an independent risk factor for further adverse event unreleased to stage of hypertension.

The other predictors of future adverse events like elevated systemic blood pressure, ejection fraction, fractional shortening are less sensitive. Regression ,of left ventricular hypertrophy occurs with treatment without deterioration in left ventricular performance. So there is a need to detect cardiac dysfunction in hypertensive patients as early as possible.

Non-invasive investigations that would provide an assessment of left ventricular status are : chest X-ray, ECG, Radio nuclide ventriculography and echocardiography. Angiocardiography is an accurate method of LVH assessment but its invasive nature and potentiality of complications, does not support its use in relatively benign condition like hypertension for left ventricular function estimation. Echocardiography provides a simple, safe, reproducible and accurate method and modality of choice to define left ventricular hypertrophy and dysfunction.

An early detection and prevention of LV dysfunction is an important goal in the management of hypertensive patient. A number of anti-hypertensive agents are available which not only controls BP effectively but also regress/reduces the risk factors associated with cardiac dysfunction.

Review

Of

Literature

REVIEW OF LITERATURE

The end of natural history of untreated hypertension is an increased likelihood of premature disability or death from cardiovascular diseases. The pathogenesis of hypertension involve structural changes in the resistance arterioles described under term remodeling and hypertrophy. Hypertrophic remodeling clearly develops in larger arteries as an early manifestations of essential hypertension (Tice et al, 1996), with close symmetry between vascular and cardiac hypertrophy (Romon et al, 2000; Vaudo et al, 2000).

Diastolic and systolic dysfunction have been observed early in the course of hypertension and either or both may lead to heart failure. Such diastolic dysfunction may reflect more vigorous atrial emptying (Ahmed et al) or abnormal diastolic relaxation (de Simone et al, 2000). The earliest function cardiac changes in hypertension are in left ventricular diastolic function, with lower E/A ratio and longer isovolemic relaxation time (Aeshbacher BC, Hutter D; Fuhreur J; et al : Am J Hypertension 14: 106, 2001).

Left ventricular hypertrophy (LVH) is detectable in 25%, 35% of all hypertensive patients and in 1% to 9% of normotensive

individuals. When present concomitant to hypertension, LVH is initially a useful compensatory process that represents an adaptation to increased ventricular stress. However, LVH is also the first step toward to development of overt clinical disease such as congestive heart failure, cardiac dysrythmias and ischemic heart disease.

Current standard for defining and diagnosing hypertension rests on blood pressure levels which confers an increased risk of developing a morbid cardiovascular event and/or will clearly benefit from medial therapy.

In adults according to VIIth report of joint national committee for prevention, detection, evaluation and treatment of high blood pressure the following values are now considered.

Systolic blood pressure :

Below 120 mmHg	Normal
120 mmHg - 139 mmHg	Prehypertension
140 mmHg – 159 mmHg	Hypertension, Stage 1
160 mmHg or above	Hypertension, Stage 2

Diastolic blood pressure :

Below 80 mmHg	Normal
80 mmHg - 89 mmHg	Prehypertension
90 mmHg - 99 mmHg	Hypertension, Stage 1
100 mmHg or above	Hypertension, Stage 2

Blood pressure was measured on two separate occasions under the proposed near ideal condition before labeling a patient as hypertensive.

Values of systolic or diastolic BP whichever was higher pressure was taken for staging.

Diastolic dysfunction precedes systolic dysfunction in hypertensive patients

Earliest functional cardiac changes in hypertension are in left ventricular diastolic dysfunction, with prolongation and in co-ordination of isovolumic relaxation time and lower E/A ratio (Braunwald 967, Dibello V. et al, 1999).

Arterial hypertension associated to LV concentric remodeling is the determinant of diastolic dysfunction but several other cardiac diseases, including myocardial ischaemia, and extra cardiac

pathologies involving the heart are other possible cause (Mausizio Cralderisi, Cardiovascular ultrasound 2005, 3:9).

Systolic cardiac function at rest is usually preserved in hypertension, however, diastolic function may be frequently altered (Nogureira JB, Acta Med Port, 1992 May; 5(5) : 269-73.

Mechanism of L.V.H. in systemic hypertension

Pathogenesis of LVH involve a number of variables other than the pressure load, one of which is hemodynamic volume load.

When the heart faces a hemodynamic overload, the major compensation is an increase in muscle mass (Lorell and Carbello, 2000).

LV mass has been found to be more closely related to systolic than to diastolic BP; the opposite is true for LV wall thickness (Schmieder and Messerli 2000).

Neurohormonal responses involving both the sympathetic and rennin angiotensin systems may be recruited to increase contractility and participate in hypertrophic response. Aldosterone increases collagen content and thereby influences adaptation and structural remodeling, independent of BP levels (Weber et al, 1994). Evidence for a critical role of the rennin-angiotensin system

is the close correlation of their circulating levels of LV mass (Harrap et al, 1996; Schmieder et al, 1996).

Different patterns were found by echocardiography in hypertensive patients 19% had normal geometry; 11% concentric remodeling; 47%, eccentric hypertrophy; and 23%, concentric hypertrophy (Wachtel et al, 2001).

Relationship between L.V.H. and L.V. dysfunction in patients of systemic hypertension

Bonaduce et al (1989) studied 13 normotensive subjects (Group I) 12 hypertensive patients without LVH (Group II) 28 with LVH (Group III). In group III patients diastolic filling parameters were impaired while in group II they were intermediate between group I and III.

Systolic dysfunction, however does not correlate well with LVH in hypertensive cases. Toshima et al reported a normal echocardiographic ejection fraction in 11 patients with concentric LVH caused by hypertension.

L.V. dysfunction in hypertensive patients with CHF

Heart failure is an abnormality of cardiac function responsible for the inability of the heart to pump blood at a rate commensurate

with the requirements of the metabolizing tissue and/or can do only from an abnormally elevated filling pressure.

Abnormalities during systole and/or diastole may be present in heart failure and/or diastole may be present in heart failure (Vasan RS et al, 1996).

In so called systolic heart failure i.e. classical heart failure, an impaired inotropic state causes weakened systolic contraction which leads, ultimately to a reduction in stroke volume, inadequate ventricular diastolic pressure. Traditionally, CHF consequent to hypertension and was considered to have only systolic dysfunction. In diastolic heart failure the principal abnormality involves impaired relaxation of the ventricle and or normal diastolic volume. Failure of relaxation can be caused by a stiffened thickened ventricle as in hypertension. So hypertension is one of the commonest cause of diastolic dysfunction and heart failure.

Assessment of left ventricular hypertrophy or enlargement :

In X-Ray cardiothoracic ratio is normally below 50% in PA view but in AP films normal value can be assessed as 55%.

In infants the normal value can be 55%. As the left ventricle enlarges, there is usually an increase in cardiothoracic ratio and

curvature of lower left heart border takes on large radius ventricles, ventricle enlarges towards lateral wall of thorax in a downward direction displacing apex laterally and inferiorly.

In lateral view we calculate distance from posterior aspect of inferior vena cava to the posterior border of heart horizontally at the level 2 cm above intersection of the diaphragm and the inferior vena cava, this is known as ***Hofman sign***. A distance of greater than 1.8 cm indicate left ventricular enlargement. Such measurements can be helpful but great reliance cannot be placed on them as individual anatomic variation can cause discrepancies (David Sutton).

In ECG for LVH detection Ramhilt and Ester point score system was used. Criteria are as follows :

			Points
1.	R or S Limb lead	: ≥ 20mm	3
	SV_1 or SV_2	: ≥ 30mm	
	RV_5 or RV_6	: ≥ 30mm	
2.	Intrinsicoid deflection in V_5 or V_6 0.05 sec or more		1
3.	Left axis deviation	: 30° or more	2
4.	QRS interval 0.09 sec or more	:	3
5.	Left atrial abnormality/ enlargement	:	3

6.	St-T changes	- without digitalis	3
		- with digitalis	1

LVH is present if the total score is more than 5 points and probably present if score is 4 points.

In echocardiography LV mass was calculated by using formula given by Devereux and Reixhek 1977 :

$$LVH = 1.04\,[(IVST+LVID+PNT)^3 - LVID^3] - 13.6$$

LV mass index is LV mass per square meter body surface area. LV mass could also be calculated from 2D echo tracing of parasternal short axis showing LV at the papillary muscle level, showing good endocardial definition by area length method (Schiller N et al 1989).

The upper limits of IVST, PWT and LV mass index (gm/m^2) are shown to be 1.1cm, 1.1cm and 122gm/m^2 BSA for Indian women (Trivedi et al 1991). In a study Ghanem Wisam MA et al 2000 LVH was defined by echocardiography as LBV mass index >134 gm/m^2 in men and >100 gm/m^2 in women.

Aims

&

Objectives

AIMS AND OBJECTIVES

⇒ *Echocardiographic evaluation of cardiac status in patients of systemic hypertension*

Material

&

Methods

MATERIAL AND METHODS

Patients of systemic hypertension were taken from Medical OPD or cardiac/Hypertension clinic.

METHODS

Patient's age, sex, occupational history, History of alcohol intake, smoking, tobacco, gutka chewing and other diversity habits. History of diabetes, obesity, angina, dyspnoea, headache, myocardial infarction, weight loss, hematuria, drug history other relevant history will be taken patient's family history of hypertension, CAD, DM, obesity was taken.

Patient's complete examination was followed by routine investigations e.g. blood sugar, blood urea, serum creatinnie, Hb, CBC, urine routine and microscopy, X-ray chest, 12 lead ECG and lipid profile.

All patients of systemic hypertension whose diastolic blood pressure was persistently over 90mm Hg, were selected for study.

But hypertensive patients with coexistent ischemic heart disease, congenital and acquired heart disease, corpulmonale, pregnancy with toxaemia, hyperdynamic circulatory states, chronic

renal failure and cardiomyopathies were excluded, as these on their own accord, are likely to alter the function of the heart.

METHODS

All the selected patient's were subjected to :-

Clinical history and examination – All cases were subjected to detailed clinical history and physical examination.

Investigations – All cases in this study were subjected to the following investigations.

1. Blood - Complete Hemogram
2. Urine - Routine and microscopic examination 24hr urinary protein
3. ECG - Standard 12 lead ECG
4. X-ray chest - PA view
5. Fundus examination

Specialized investigation

1. Blood sugar Fasting Post prandial
2. Blood urea
3. Serum creatinine

4. Serum electrolytes (Na^+, K^+)

5. Lipid profile : Total cholesterol, LDL, HDL, triglyceride (12-14 hours fasting

6. Abdominal ultrasonography

7. Echocardiography

All selected patients will be subjected to echocardiography.

The left ventricular dysfunction was assessed in the following heading –

1) LVH

2) Diastolic dysfunction

3) Systolic dysfunction

LVH : There are two methods of calculating LV mass from 2D echocardiography.

a) Area length method

b) Truncated ellipsoid method

For both methods require short axis view of left ventricle at papillary muscle level and apical four or two chamber at end

diastole are required. Myocardial mass is equal to product of volume and specific gravity of myocardium (1.04 gm/ml).

Built in software in ultrasound can make both methods available so that mass is automatically calculated, once all variables are fed. LV mass can also be estimated from 2D guided, M mode measurements of LV dimension and wall thickness at papillary muscle level without measuring left ventricular major axis. Dimension and simple geometric cube formula. The following equation provides an accurate determination of LV mass, according to Devereux and associated –

Left ventricular mass (gms)

$$= 1.04\ (\mathrm{LVID} + \mathrm{PWT} + \mathrm{IVST})^3 - \mathrm{LVID}^3 \times 0.8 + 0.6$$

where

1.04 specific gravity of myocardium

0.8 correction factor

LVID – Left ventricular internal dimension

PWT – Posterior wall thickness

IVST – Interventricular sept. thickness measured at end diastole

Diastolic dysfunction

Based on Doppler velocity pattern, diastole dysfunction is divided into three categories –

a) Relaxation abnormalities

b) Restrictive physiology

c) Pseudonormalization

Relaxation abnormalities

Abnormal myocardial relaxation characterized by constellation of following abnormalities

Prolonged IVRT (Isovolumic relaxation time) > 110 m/sec

Low E velocity (early filling velocity) and high

A velocity (A velocity = Late filling velocity)

Revised E/A ratio (<1.0)

Prolonged deceleration time (DT) > 240 m/sec

Restrictive physiology : is characterized by following diastolic parameters

Shortened IVRT (<60 msec)

High E velocity and low velocity

Increased E/A ratio > 2

Shortened deceleration time (< 150 msec)

Systolic dysfunction

To evaluate systolic two parameters are used :

1) Fractional shortening or ejection fraction

2) Cardiac output

Fractional shortening : is a percentage change in left ventricle cavity dimension with systolic contraction and can be calculated from following equation.

Fractional shortening = LVED-LVES) / LVED × 100%

Where

LVES – LV end systolic dimension

LVED – LV end diastolic dimension

Ejection fraction : Represents stroke volume as percent of end diastolic left ventricular volume.

Ejection fraction = (EDV – ESV) / E ×100%

Where

EDV - End diastolic volume of LV

ESV - End systolic volume of LV

Quninomers and co authors proposed a simplified method for determination of ejection fraction by measuring LV internal dimensions

$$\text{Ejection fraction} = (\%\triangle D^2) + [C1 - \%\ D\triangle^2]\ [\%\triangle L]$$

Where

$$\%\triangle D^2 = [(LVED - LVES) / LVED^2] \times 100\%$$

Observations

OBSERVATION

The present study was undertaken in 60 hypertensive patients of different age groups severity and duration of hypertension to assess LV systolic and diastolic functions by means of echocardiography. The findings are as follows :

Table -1 : *Age and Sex distribution of hypertensive cases*

S. No.	Age	Male	Female	Total	Percentage
1.	31-40	10	2	12	20.00
2.	41-50	14	6	20	33.33
3.	51-60	14	6	20	33.33
4.	61-70	2	6	8	13.33
	Total	**40**	**20**	**60**	**100.00**

Table shows that out of 60 patients, 40 (66.66%) were male, and 20 (33.33%) were females.

Maximum number of patients belonged to 41-60 years. Mean age was 49.3 years.

Table -2: Prevalence of left ventricular dysfunction in hypertensive patients with or without congestive heart failure (CHF)

S. No.	Dysfunction	No. of cases (60)	Percentage
1.	Only diastolic dysfunction	38	63.3
2.	Only systolic dysfunction	2	3.3
3.	Both systolic & diastolic dysfunction	6	10
4.	No LV dysfunction (either systolic or diastolic)	14	23.3
5.	Total diastolic dysfunction (alone diastolic or with systolic dysfunction)	46	73.3
6.	Total systolic dysfunction	8	13.3

Table 2 shows that LV dysfunction was present in 76.6% of hypertensive patients. Among 46 cases of LV dysfunction 73.3% had diastolic dysfunction and 13.3% had systolic dysfunction. 10% had both systolic and diastolic dysfunction and 63.3% had only diastolic dysfunction, and those with systolic dysfunction was only 3.3%.

Table -3: **Incidence of LV dysfunction in hypertensive patients with clinically diagnosed CHF.**

S. No.	Dysfunction	No. of cases (6)	Percentage
1.	Only diastolic	2	33.3
2.	Only systolic	2	33.3
3.	Both	2	33.3.

Out of 60 cases of hypertension 10% patients i.e. 6 of them presented with CHF. Among them LV diastolic dysfunction, systolic dysfunction and both dysfunction was equally prevalent 33.3%.

Table -4: **Spectrum of echocardiographic findings in patients with diastolic dysfunction only**

S. No.	Parameter	Cases with diastolic dysfunction only (n=38)	Percentage	Total cases with diastolic dysfunction (n=44)	Percentage
1.	Reduced EF slop	38	100	44	100
2.	Altered E/A	38	100	44	100

EF slope was reduced and E/A ratio altered in all the cases with diastolic dysfunction.

Table -5: Spectrum of echocardiographic findings in patients with Systolic dysfunction only

S. No.	Parameter	Cases with systolic dysfunction only (n=2)	Percentage	Total cases with systolic dysfunction (n=44)	Percentage
1.	Reduced LVEF	2	100	8	100
2.	Increased LVIDd	2	100	8	100
3.	Increased LVIDs	2	100	8	100

Increased LV dimension and reduced ejection fraction was present in all the cases of systolic dysfunction.

Table -6: Comparison of LVH detected by X-ray, ECG and echocardiography among 60 cases of hypertension

S. No.	Method	Hypertensive cases with LVH	Percentage
1.	Chest X-ray	2	3.33
2.	ECG	4	6.66
3.	Echocardiography	32	53.3

By echocardiography, LVH was detected in 53.3% of hypertensives whereas ECG could detect it only in 6.66% and chest radiography in 3.3% cases of hypertension.

Table -7: Relationship of LV functions with LVH in cases of hypertension

S. No.	Dysfunction	Total no. of cases (n=60)	No. of cases with LVH (n=32)	Percentage
1.	Only diastolic	38	26	68.40
2.	Only systolic	2	2	100.00
3.	Both	6	4	66.60
4.	Total diastolic	44	30	68.18
5.	Total systolic	8	6	75.00
6.	Both dysfunction with CHF	2	2	100.00
7.	Both dysfunction without CHF	4	2	50.00

Prevalence of LVH in hypertensive cases is as follows. In only diastolic dysfunction cases 68.4%. In patients with systolic dysfunction 100%, 66.6% in patients with both dysfunction. No LVH in hypertensive patients with normal LV function in 68.18% in total patients with diastolic dysfunction. In 75% of total systolic dysfunction.

Table -8: Correlation between LV dysfunction and duration of hypertension

S. No.	Duration in yrs	No. of hypertensive cases (n=60)	No. of hypertensive cases with diastolic dysfunction (n=44)	Percentage	Hypertensive cases with systolic dysfunction (n=8)	Percentage
1.	<5	8	14	50	4	14.28
2.	5-10	20	18	90	-	-
3.	>10	12	12	100	4	33.3

In cases with less than five years duration of hypertension, 50% had diastolic dysfunction and 14.28% had systolic dysfunction.

In cases with 5-10 years, none had systolic whereas 90% had diastolic dysfunction.

In more than 10years duration, all had diastolic dysfunction and 33.3% had systolic dysfunction.

Table -9: LV dysfunction in hypertensive patients of different age groups

S. No.	Age group (in yrs)	No. of hypertensive cases (n=60)	No. of hypertensive cases with diastolic dysfunction (n=44)	Percentage	Hypertensive cases with systolic dysfunction (n=8)	Percentage
1.	31-40	12	6	50	2	16.6
2.	41-50	20	14	70	-	-
3.	51-60	20	16	80	6	30
4.	61-70	8	8	100	-	-

Systolic dysfunction was present in 16.6% in 4th decade, 30% in 6th decade. Diastolic dysfunction was present in 50% in 4th decade and increased with age till 100% in 7th decade.

Table -10: LVH in hypertensive patients of different age groups

S. No.	Age group (In years)	Hypertensive cases (n=60)	Hypertensive cases with LVH (n=32)	Percentage
1.	31 – 40	12	4	33.3
2.	41 – 50	20	10	50
3.	51 – 60	20	10	50
4.	61 – 70	8	8	100

33.3% cases had LVH among hypertensive in 4th decade. In 5th and 6th decade LVH was relevant equally i.e. in 50% cases. All the patients had LVH in 7th decade.

Table -11: Correlation between LVH and duration of hypertension

S. No.	Duration in years	Hypertensive cases (n=60)	Hypertensive cases with LVH	Percentage
1.	5	28	8	28.5
2.	5 – 10	20	12	60
3.	10	12	12	100

LVH was present in 28.5% cases with hypertension of less than five years duration. It increased to 60% in cases with 5 – 10 years duration and all the cases has LVH in hypertension of more than 10 years.

Table -12: Correlation between severity of hypertension and cardiac dysfunction

S. No.	Severity of hypertension	Hypertensive cases (n=60)	Diastolic dysfunction cases (n=44)	Percentage	Systolic dysfunction cases (n=8)	Percentage
1.	Prehypertension	24	18	75	2	8.3
2.	Stage I	20	14	70	-	-
3.	Stage II	16	12	75	6	37.5

75% had diastolic dysfunction and only 8.3% had systolic dysfunction in persons with prehypertensive group. There was no systolic dysfunction in the stage I group only diastolic dysfunction in 70% cases. In patients with stage II hypertension 37.5% had systolic dysfunction an 75% had diastolic dysfunction.

Table -13: Correlation between severity of hypertension and LVH

S. No.	Severity of hypertension	Hypertensive cases	Hypertensive cases with LVH	Percentage
1.	Prehypertension	24	10	41.6
2.	Stage I	20	10	50
3.	Stage II	16	12	75

In patients with prehypertension, LVH was present in 41.6%. This increased to 50% in stage I an 75% in stage II hypertension.

Table -14: Sex wise distribution of cases of LVH and LV dysfunction

S. No.	Hypertensive cases	Male (n=40)	Percentage	Female	Percentage
1.	LVH	20	50	12	60
2.	Cases with diastolic dysfunction	28	70	16	80
3.	Cases with systolic dysfunction	8	20	-	-

Hypertensive females presented with LVH in 60% and diastolic dysfunction in 80%. Male had LVH in 50%, diastolic dysfunction in 70% and systolic dysfunction in 20%.

Table -15: Correlation between LVH and LV dysfunction and control of blood pressure

S. No.	Cases	BP control satisfactory (n=32)	Percentage	Bp unsatisfactory (n=28)	Percentage
1.	LVH cases	12	37.5	20	71.4
2.	Cases with diastolic dysfunction	12	68.75	22	78.5
3.	Cases with systolic dysfunction	2	6.25	6	21.42

Hypertensives with satisfactory control of BP had LVH and systolic dysfunction in 6.25%. Those with unsatisfactory BP control had LVH in 71.4% diastolic dysfunction in 78.5% and systolic in 21.42%.

Discussion

DISCUSSION

The present study was undertaken to asses echocardiographic evaluation of systemic hypertensive patients by means of echocardiography (M-mode, 2D and Doppler) in 60 cases.

Although conventional ECG and radiography provide some information about the cardiac anatomy, these remain silent regarding myocardial functional impairment. Contrast ventriculography in addition to being a invasive investigation carries risk of nephropathy, anaphylaxis and more over because of dye, diuresis may influence ventricular function.

Out of these we had chosen echocardiography as this can be employed safely in any setting without patient preparation, discomfort and convinence. This is noninvasive economical and can be repeated any number of times.

The age wise distribution of hypertensive cases in various age group has been well highlighted by many workers like Janeway (1913), Bell and Clawson (1928), Be Chgaard (1946) and Paulwood (1968). In our study, the age of patients with systemic hypertension ranged from 31-68 years with mean of 49.3±9.8.

maximum number of cases were between 41-60 years being equally divided in 5th and 6th decades.

The male : female ratio of 2:1 in conformity with the study of Bell and Clawson (1928) and Gaur et al (1956).

Echocardiography vs. ECG and radiography to detect LVH in systemic hypertension

Echocardiography showed LVH in 53.3% cases of hypertension as given in table-6. In these patients ECG indicated LVH in only 2 cases (6.6%) while radiography was even a poorer parameter which suggested LVH in only one case (3.3%). Echocardiography therefore, was found to be far superior method to detect earliest increase in the girth of LV wall. Early detection may useful in predicting not only the prognosis but also highlighting therapeutic measure to control hypertension.

In the study of Savage et al (1989), 3% cases were found to have LVH in hypertensives and 5% by chest X-ray while echocardiography detected in 67%. Pearson, Gudipati et al (1987), found that out of 15 patients of hypertension, only one met ECG criteria of LVH, while 8 cases have echocardiographically proved LVH. Findings are also in conformity with Woythaler JN et al

(1983) and Daugherty et al (1984), who found quantitative M-mode echocardiography more sensitive and specific as compared with electrocardiographic voltage and Este's criteria as an index of hypertrophy.

LVH and LV dysfunction

In the present study LV dysfunction was found in 76.6% hypertensive subjects while it was normal in rest 23.3% Savage et al (1989), found deranged LV function in 60% cases of hypertension.

Among the hypertensive cases 73.3% had diastolic dysfunction while only 13.3% had abnormal systolic function. Combined dysfunction was found in only 10% cases. These findings are consistently with so many workers who also found systolic dysfunction uncommon in such cases while diastolic dysfunction was the rule (Pearson and Labovitz, 1987).

The incidence of LVH was 53.3% in this study by echocardiography, among cases of systemic hypertension. Similar incidence is reported by Savage et al (1989), who found it in 615 cases of hypertension by echocardiographic measurements. All the cases with LVH had LV dysfunction and that too mostly in the

form of diastolic dysfunction (100%), while systolic or combined dysfunction occurred in only 18% cases of LVH. In patients with combined dysfunction if LVH it was present, it was of eccentric type.

This also is in agreement with various authors. Smith et al (1985), Pearson and Labovitz (1987), and Papademetriou et al (1985), found abnormalities in diastolic properties of left ventricle in patients with LVH secondary to systemic hypertension, an usual finding.

In the present study diastolic dysfunction was present in 6 cases without LVH and one case of systolic dysfunction was present without LVH among hypertensives. Inouye et al (1984) had also observed LV diastolic dysfunction without LVH. The occurrence of diastolic dysfunction preceding LVH could be explained on the basis that factors beside increase in LV mass are also responsible for altered LV filling dynamics. The other possibility is that in such patients although increase in LV mass has occurred but yet this increase is not sufficient enough to meet criteria for LVH by echocardiography.

Inouye et al (1985) also observed that severity of LV diastolic dysfunction increases in hypertensives with LVH.

Risk factors and LV dysfunction

In the present study LV dysfunction correlated well with advancing age of patient. This is in agreement with the study of Savage et al (1989) who also found decrease in ejection fraction with increase in age.

No definite correlation was found between severity of hypertension on LV dysfunction in this study which is consistent with conclusions of Savage et al (1989).

LVH in hypertensive patients increases with increase in age, duration and severity in the present study.

Savage et al (1989), also found positive correlation with age of hypertensive and severity and duration of hypertension.

In this study there was positive correlation of LV dysfunction with increase in age and blood pressure.

LV dysfunction was present in asymptomatic cases of hypertension also which is important from therapeutic point of view for other wise these cases would have been treated as without

target organ damage. No sex wise preponderance of LV dysfunction was found as in Inouye et al 1985 and others.

CHF and Systemic hypertension

Congestive heart failure is a common and often lethal complication of chronic systemic hypertension. In the present study 3 cases of systemic hypertension also had CHF clinically. On echocardiographic evaluation, diastolic dysfunction, systolic dysfunction, and combined dysfunction was found equally prevalent in them i.e. 33%.

Earlier studies implicated that in CHF, only systolic functions of LV was deranged, but now CHF cases with isolated diastolic or combined dysfunction has been recognized by Pearson and Labovitz (1987), Topol et al (1990). Among such CHF cases with diastolic dysfunction, systemic hypertension has been found to be a major cause.

Inouye et al (1984), postulated that systolic dysfunction is uncommon in patents with prehypertension and stage I hypertension, even though this group forms the substrate form which many persons with CHF will emerge. In contrast, diastolic dysfunction is the rule.

This is consistent with the early appearance of left atrial enlargement in the evolution of LVH and may explain the dyspnoea and pulmonary congestion, which are sometimes seen in hypertensive patients.

Our findings are in agreement with those of Dougherty et al (1984), who studied 188 patients with CHF. 64% of them had reduced LVEF while 36% had normal EF. Of these patients with normal EF, 65% were due to systemic hypertension.

These finding point out that such patients need little inotropic support, rather therapy should be aimed at improving the diastolic filling.

Echocardiography thus permits rapid and non invasive detection of cardiovascular complications in systemic hypertension, some of which would remain unrecognized or even unsuspected.

Conclusion

&

Summary

CONCLUSION AND SUMMARY

The following conclusion emerge form the present study :

1. The age of our patients of systemic hypertension ranges from 31-68 years with mean of 49.3 ± 9.8.
2. Maximum number of cases fall between 41 and 60 years, being equally divided in 5^{th} and 6^{th} decades.
3. Male – female ratio is 2:1 in hypertensive individuals.
4. LV dysfunction is present in 76.6% of hypertensive patients while, it remains normal in remaining 23.3%. Left Ventricular dysfunction may be either systolic or diastolic or both in such patients.
5. 73.3% cases of systemic hypertension have LV diastolic dysfunction including 63.3% cases of isolated diastolic dysfunction and 10% cases of combined systolic and diastolic dysfunction.
6. Of the cases of combined LV systolic and diastolic dysfunction, 3.3% cases present with CHF whereas 6.6% have no evidence of CHF.
7. CHF complicate hypertension in 10% cases.

8. in patients of CHF complicating hypertension, isolated LV systolic dysfunction, isolated diastolic dysfunction and combined LV diastolic and systolic dysfunction is present in equal number of cases (33% in each group).

9. Echocardiographic parameters in cases of systemic hypertension with diastolic dysfunction are : Reduced EF slope and altered E/A ratio in all the cases (100%).

10. Echocardiographic parameters in cases of systemic hypertension with systolic dysfunction are : Reduced LVEF and increased LVID in all the cases (100%).

11. Echocardiographic evidence of LVH occurred in 53.3% cases of systemic hypertension. While LVH occurred in 68.4% cases of systemic hypertension on presenting with diastolic dysfunction. Isolated systolic dysfunction is also associated with LVH.

    All the cases with LVH show LV dysfunction either systolic or diastolic or combined.

    Diastolic dysfunction occurs in all the cases (100%) of systemic hypertension with LVH.

Systolic dysfunction also occurs in all cases of systolic hypertension with LVH. Both systolic and diastolic dysfunction can occur without LVH.

12. There is an increase in the incidence of LV diastolic dysfunction with increasing duration of systemic hypertension, but statistically, the increase in duration was not significant. There is no correlation between increase in duration of hypertension and systolic dysfunction.

13. LV diastolic dysfunction is also usually related to the age of patient, as this increases with advancing age irrespective of the duration of hypertension; but systolic dysfunction has no correlation.

14. Incidence of LVH usually increases with advancing age in hypertensive cases being 33.3% in 4^{th} decade; 50% in 5^{th} and 6^{th} decades and 100% in 7^{th} decades.

15. Duration of hypertension is proportional to LVH and is found to be higher with increase in duration. The increase in duration in patients with LVH is statistically significant ($p<0.01$, $t=2.9$).

16. There is no definite correlation between severity of hypertension and cardiac dysfunction although, maximum number of cases of systolic dysfunction occurs in patients with stage II hypertension.

17. Severity of hypertension is related to LV wall thickness as the incidence of latter increase with severity of hypertension being, 41.6% in prehypertension, 50% in stage I and 75% in stage II cases.

18. LVH and diastolic dysfunction is slightly more in female hypertensives whereas systolic dysfunction is present in males.

19. Hypertensive patients with satisfactory control of blood pressure have lesser complication like LVH and LV dysfunction, compared to those with poor control.

20. LV diastolic and systolic dysfunction is equally prevalent between symptomatic and asymptomatic groups of hypertensives.

21. LVH could be detected in 3.3% and 6.6% cases by chest radiograph and ECG respectively while echocardiography could detect it n 53.3% cases of hypertension. Thus

echocardiography is far superior laboratory tool for LVH detection compared to convention X-ray and ECG.

22. Assessment of systolic and diastolic dysfunction in cases of CHF complicating systemic hypertension has great clinical importance. In these patients because medications such as digitalis, vasodilators and diuretics, which are commonly employed to treat CHF, suspecting only systolic dysfunction, may in fact have an untoward effect on patients, with coexistent or isolated diastolic dysfunction. While medications with known negative inotrophic effects such as calcium channel blockers and β blockers may prove beneficial.

23. Echocardiography provides a reliable, noninvasive and sensitive method for detecting accurately LVH and LV systolic and diastolic dysfunction, which have important diagnostic, therapeutic and prognostic implications in patients of systemic hypertension.

Appendix

APPENDIX

A	Mitral peak flow velocity during atrial contraction
CHF	Congestive heart failure
E	Mitral peak flow velocity during rapid filling phase
LVEF	Ejection fraction of left ventricle
IVSd	Inter ventricular septal thickness at end diastole
IVSs	Inter ventricular septal thickness at end systole
LVIDd	Left ventricular Internal dimension at end diastole
LVIDs	Left ventricular Internal dimension at end systole
LVH	Left ventricular hypertrophy
LVPWd	Left ventricular posterior wall thickness at end diastole
LVPWs	Left ventricular posterior wall thickness at end Systole

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